2:30

755-3 Spectral Turbulence Versus Time-Domain Analysis of the Signal-Averaged ECG for Risk Stratification After Myocardial Infarction

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Spectral turbulence analysis (STA) of the signal-averaged ECG (SAECG) combines spectral analysis with a statistical evaluation of spectrograms in individual parts of the QRS complex. It has been suggested that it may be superior to conventional time-domain analysis (TDA) of the SAECG.

Methods: We compared the power of TDA (40–250 Hz) and STA of the SAECG for prediction of cardiac death, ventricular tachycardia, sudden arrhythmic death, and arrhythmic events after myocardial infarction in 603 patients. The population excluded patients with bundle branch block and other conduction abnormalities.

Results: During the first 2 years of follow-up, there were 40 cardiac deaths, 21 ventricular tachycardia, 11 sudden arrhythmic deaths, and 29 arrhythmic events. The positive predictive accuracy of STA was significantly higher than TDA for the prediction of cardiac death at most sensitivity levels (26% vs 20%, respectively, at 40% sensitivity, $p < 0.05$). The positive predictive accuracies were not statistically different for the prediction of ventricular tachycardia. For the prediction of sudden arrhythmic death and arrhythmic events, the positive predictive accuracy of STA was better than that of TDA only at high sensitivity levels (respectively 9% vs 2%, $p < 0.001$ for sudden arrhythmic death; and 14% vs 11%, $p < 0.05$ for arrhythmic events; at 60% sensitivity).

Conclusion: STA is essentially equivalent to time-domain analysis for the prediction of arrhythmic events after myocardial infarction. However, STA performs significantly better than TDA for the prediction of total cardiac mortality after myocardial infarction.

2:45

755-4 Dobutamine-Induced Recovery of Asynergy Improves the Abnormal Signal Averaged ECG After Myocardial Infarction

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After myocardial infarction (MI) viable myocardium between normal and necrotic areas may represent a possible substrate for an abnormal signal averaged ECG (SAE). Myocardial viability can be assessed by low-dose dobutamine (Dob)-Echo on the basis of the contractile recovery of a basal asynergy. Aim of this study was to assess whether Dob-induced improvement in contractility may change SAE in patients (pts) with MI. Twenty-five pts, 19 males, mean age 58 years, underwent a Dob-Echo test 13 ± 5 days after AMI. SAE (40 Hz, Butterworth filter) was recorded before and during a 6' Dob infusion (10 μg/Kg). Quantitative wall motion analysis of LV divided in 24 segments was done on digitized images and % area changes (%AC) from peak diastolic to peak systolic area calculated by centerline method. Asynergy was found in 111/600 segments (basal %AC < 30%); segments were considered viable when %AC increased by ≥ 35% from baseline. Dob improved LV contractility in 13 pts (%AC from 26 ± 5 to 41 ± 4) [G I]; in 12 pts [G II] no changes in %AC were observed (from 24 ± 4 to 24 ± 6%). Site of MI, thrombolysis, basal EF, peak heart rate at 10 μg/Kg Dob were comparable in both groups; a baseline (B) significantly ($p < 0.01$) longer fQRS and LAS40 and shorter RMS40 were recorded in G I pts. Shortening of fQRS and prolongation of RMS40 were seen in pts with Dob-induced contractile improvement; in pts with no myocardial viability these parameters had an opposite behaviour.

	fQRS		RMS40		LAS40	
	B	Dob	B	Dob	B	Dob
G I	113 ± 10	108 ± 8**	20.6 ± 6	30 ± 10*	30.4 ± 10	30 ± 10
G II	101 ± 10	110 ± 11*	35.8 ± 13	24 ± 10**	23.8 ± 8	31 ± 14

*$p < 0.05$, **$p < 0.01$ (B vs Dob)

Dob-induced contractile recovery of asynergic areas parallels an improvement in SAE parameters which is likely due to a "homogenizing effect" on the myocardium with reduction in delay and fragmentation of ECG activation fronts.

3:00

755-5 The Influence of Infarct Healing on Quantitative Waveform Features of Cardiac Late Potentials

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Controversy exists over the influence of infarct (MI) healing on cardiac late potentials (LP) identified by body surface signal averaged ECG (SAECG). Inherent methodological limitations associated with SAECG contribute to this controversy. The purpose of this investigation was to quantitate the waveform characteristics of cardiac LP electrograms (EGM) recorded directly from infarcted myocardium during MI healing. The study was performed in a closed chest canine model (n = 16) with a healing anterior wall MI, an indwelling myocardial electrode array and inducible sustained ventricular tachyarrhythmia (VT). Orthogonal LP EGMs were mapped, amplified and digitized at weeks 1, 4 and 8 of infarct healing. Single beat time-frequency analysis of the digitized EGMs was performed to quantitate the energy of 3 different frequency bands ([10–50 Hz] — low; [51–160]) Hz] — mid; [161–300 Hz] — high). *Results:* Compared to controls, LP EGMs showed significant increases ($p < 0.05$) in energy during healing. Increases in energy in the high frequency band were observed more often in the terminal portions of the EGM while energy increases in the low and mid-frequency bands were scattered throughout the duration of the EGM. No significant change occurred in LP EGM duration with MI healing. *Conclusion:* Infarct healing is associated with increases in the energy content but not duration of LP EGMs. These observations may help to explain some of the variability in SAECG recorded in patients during infarct healing.

3:15

755-6 Lack of Stability of Late Potentials During the First Year After Myocardial Infarction in the Thrombolytic Era

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In survivors of acute myocardial infarction (MI) in the thrombolytic era, the stability of late potentials (LP) detected in the signal-averaged ECG (SAECG) has not been examined in detail. Thus, 2 SAECGs were recorded, the first (SAECG-1) 10 days, the second SAECG-2) 1 year post MI in 95 consecutive pts to assess the stability of LP. To assess the role of recurrent myocardial ischemia in provoking changes in the SAECG, 7 clinical factors (age, sex, infarct location, recurrent myocardial infarction or ischemia, performance of PTCA or CABG) were examined.

After 1 year, the results of the 2 SAECG recordings were identical in 75/95 pts (79%). Nine pts showed a new LP on SAECG-2 whereas 11 pts with an initial positive SAECG had lost their LP after 1 year. Of the 9 pts with a new LP after 1 year, 1 had recurrent ischemia compared to 23/67 remaining negative at SAECG-2 ($p = 0.3$). From the 11 pts who had lost their LP on SAECG-2, 4 had recurrent ischemia compared to 1/8 with a positive SAECG-2 ($p = 0.52$). From the 28 pts with LP either on SAECG-1 or SAECG-2, only 1 developed an episode of sustained VT during follow-up.

Thus, in post-MI pts in the thrombolytic era, there is a considerable change in the results of SAECG recordings acutely and after 12 months. Ischemia appears not to be a decisive factor in modifying the incidence of LP. These results question the usefulness of LP as a tool for risk stratification post MI in the thrombolytic era.

756 Prolonged QT Syndrome

Tuesday, March 26, 1996, 2:00 p.m.–3:30 p.m.
Orange County Convention Center, Room 414B

2:00

756-1 Long QT Syndrome Patients Genetically Linked to Defective Genes on Chromosomes 11, 7 and 3 Present Differential Response to Changes in Heart Rate

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The genes for the long QT syndrome (LQTS) linked to chromosomes 3 (LQT3) and 7 (LQT2) were identified as SCN5A, the cardiac sodium channel gene, and as HERG, a potassium channel gene, no altered gene has been identified for LQTS linked to chromosome 11 (LQT1). Based on experimental